5-1958

Artificial Respiration during the Apnea of Cerebral Trauma

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School of Graduate Studies

ARTIFICIAL RESPIRATION DURING THE APNEA OF CEREBRAL TRAUMA

by

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A Thesis in Partial Fulfillment

of the Requirements for the Degree

Master of Science in the Field of Physiology

May, 1958

I certify that I have read this thesis and that in my opinion it is fully adequate, in scope and quality, as a thesis for the degree of Master of Science.

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ACKNOWLEDGEMENT

With great appreciation I thank:

Dr. Kenneth E. Kellogg for his advice, encouragement, and criticism in connection with the project herein reported and this paper.

Mrs. Geraldine Applegate for petience and diligence in typing this manuscript.

Mr. James Sung for valuable technical assistance.

The Los Angeles office of Baldwin-Lima-Hamilton Company for the gift of a number of SR-4 strain gages used in this project.

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INTRODUCTION

The profound respiratory depression associated with severe cranial injury has been the subject of considerable research by a number of investigators since Polis reported his classical studies on cerebral concussion in 1894.

It has been demonstrated that when a blow of sufficient force is delivered to the head of any of the commonly used laboratory animals, almost immediately a quick inspiratory gasp occurs, which is followed by a relatively slow expiratory excursion. This expiratory phase in turn merges into an apneic interval the duration of which can be generally related to the magnitude of the blow. There is also classically a concomitant arterial hypertension the magnitude and duration of which may also be correlated generally with the concussive force. The blood pressure usually reaches normotensive levels in from 1-2 minutes unless the injury is severe. When the respiratory arrest is unduly prolonged the arterial blood pressure falls to fatal levels in a fashion similar to that seen in fulminating anoxia and asphysia. Because it appears that sudden death after head injury may be due to a lack of pulmonary ventilation, several investigators have studied the beneficial effects of artificial respiration during the period of respiratory depression described above.

Kramer and Horsely (14), using dogs under ether anesthesia and insulting the brain with bullets (of presumably low velocity (15),

caliber unstated), through the cerebral hemispheres, found that death could be prevented by artificial respiration except under special circumstances, which were not stated. Kramer (15) reaffirmed this position using a falling weight and .22 caliber bullets impinging on a brass plate fastened to the skull. He considered apnea to be the most severe physiological effect of concussion. Polis (18) and Cannon (2) also agreed that artificial respiration may save life in many cases.

Denny-Brown and Russell (4), however reported that given a sufficient degree of cerebral trauma, artificial respiration during or before blood pressure failure, or even during the entire experiment, did not prevent the blood pressure from falling to and maintaining a very low level (about 10 mm Hg.). This value is too low for maintenance of adequate nourishment of central nervous system tissue. A weighted pendulum with limited travel was used to produce concussion in the work of Denny-Brown and Russell. Because of these differences of opinion, it was felt that further investigation might resolve this problem. Accordingly a series of experiments was planned in which prolonged respiratory arrest would ensue from the delivery of an adequate blow or blows to the head. The efficacy of artificial respiration could then be assessed by appropriate pulmonary ventilation when the arterial pressure had dropped precipitously to hypotensive levels incompatible with life.

METHOD

Mongrel cats averaging slightly more than two kilograms in weight were anesthetized by the intraperitoneal injection of sodium pentobarbital. It was found that an initial dose of 25-35 mg/kg produced the desired level of light surgical anesthesia. In certain instances a

somewhat smaller amount was employed in order to assure the preservation of important medullary functions which may be depressed by the more liberal use of the barbiturates. When necessary, additional sodium pentobarbital was administered.

The anesthetized cat was secured to an animal board in the customary fashion after which the trachea was cannulated. A small polyethylene catheter was placed in a superficial vein and connected to a syringe filled with isotonic saline for the intravenous infusion of the anesthetic agent, vasopressors, etc. when needed. Another small polyethylene catheter was inserted into the right femoral artery, usually, and connected to an unbonded strain gage (Statham P-23AA) designed for measuring fluid pressures. This system was perfused with isotonic saline under positive pressure by a constant rate pump which delivered 11.6 ml/hr. The perfusion pump (Figure I) was assembled in the Physiology shop and consisted of a 50 cc syringe, the plunger of which was advanced about 1.9cm/hr. by a motor driven jackscrew. This method was chosen to keep the arterial catheter free of clots in order to diminish the danger of hemorrhage which would more likely result from the conventional method of heparin infusion because of the traumatic nature of the experiment.

The transducer output was fed into a carrier preamplifier using 2400 cycles/second excitation; the amplified signal in turn having been acted upon by a linear rectifier was transferred to the pen motor circuit. This system acts as a stable direct current amplifier for the low frequencies involved. Finally arterial pressure was recorded on one channel of a series 150 Sanborn multi-channel hot stylus oscillograph.

The range and calibration as used in all experiments herein reported are as follows:

Zero suppression out 0 - 125 mm Hg. spread over 50 mm

Zero suppression in 50 - 300 mm Hg. spread over 100 mm

Zero suppression introduces a fixed (but adjustable) signal in the input of the penwriter which effectively offsets the no-signal position of the stylus to a new position, which can be off the recording paper. This does not change the basic calibration. The entire system was balanced and calibrated according to the manufacturer's instructions before each experiment.

Respirations were recorded in one of the following two ways:

A. A small bellows type pneumograph was fastened to the cat's chest and connected to an unbonded strain gage (Statham P-23BB) which recorded respiratory excursions on one channel of the oscillograph.

B. Because of certain limitations inherent in bellows type pneumographs, a bonded strain gage (Baldwin-Lima-Hamilton SR-4) was cemented to a plastic block 3/16" by 1½" by 3/4" (Figure II). This was attached to a 3/8" wide elastic material which encircled the cat's chest. The force of inspiration deformed the plastic material sufficiently to give much improvement in the respiratory recording and was sensitive enough to show a small deflection synchronous with the heart beat during apnea. Later, a more sensitive pickup using two strain gages was fabricated and used in some of the later experiments (Figure III). This device consisted of a piece of spring steel with a strain gage cemented on each side. The bonded strain gages have a frequency response to at least 50 kilocycles/sec. and are capable of showing approximately 3 mm stylus deflection with 20 microinches linear gage displacement. The circuit diagram is shown in Figure IV.

Artificial respiration was provided by an electric piston pump (Palmer) with variable stroke and cycle rate. The rate and depth was such that some hyperventilation occurred in all cases. The rate approximated that of the cat's natural breathing as seen on the respiratory record, but the depth was greater according to the respiratory record, in order to insure adequate ventilation.

The head of the cat was turned ninety degrees to the left while being hit. The concussive blow was delivered to the cat's occipit by a spring loaded hammer after the method of Gurdjian and Webster (7) with minor modifications (Figure V). The velocity of the blow and spring tension were not measured. The spring was extended so that hammer velocity was sufficient to produce prolonged apnea. The springs used developed some set and had to be replaced at intervals. It has been shown that head acceleration is not related to the degree of concussion (11). The variables of head placement, neck muscle tone, etc., make quantitative measurement of hammer velocity at impact almost meaningless. However, the velocities were on the order of seven meters per second at impact. This was estimated by the use of a pair of electromagnetic gates and the measurement of transit time using an oscilloscope. camera and a time base gnerator. The value cited is consistent with that reported by Denny-Brown and Russell (3). Gurdjian, et al (11, 12) and Haddad (13) showed that the severity of the concussion is roughly proportional to the time duration of the pressure and acceleration. It was not possible with the apparatus used, to exercise precise control over these variables, so except for stating the blow was moderate or maximum (for the hammer and spring used), an exact statement of applied trauma in each case has been omitted. The hammer was limited to

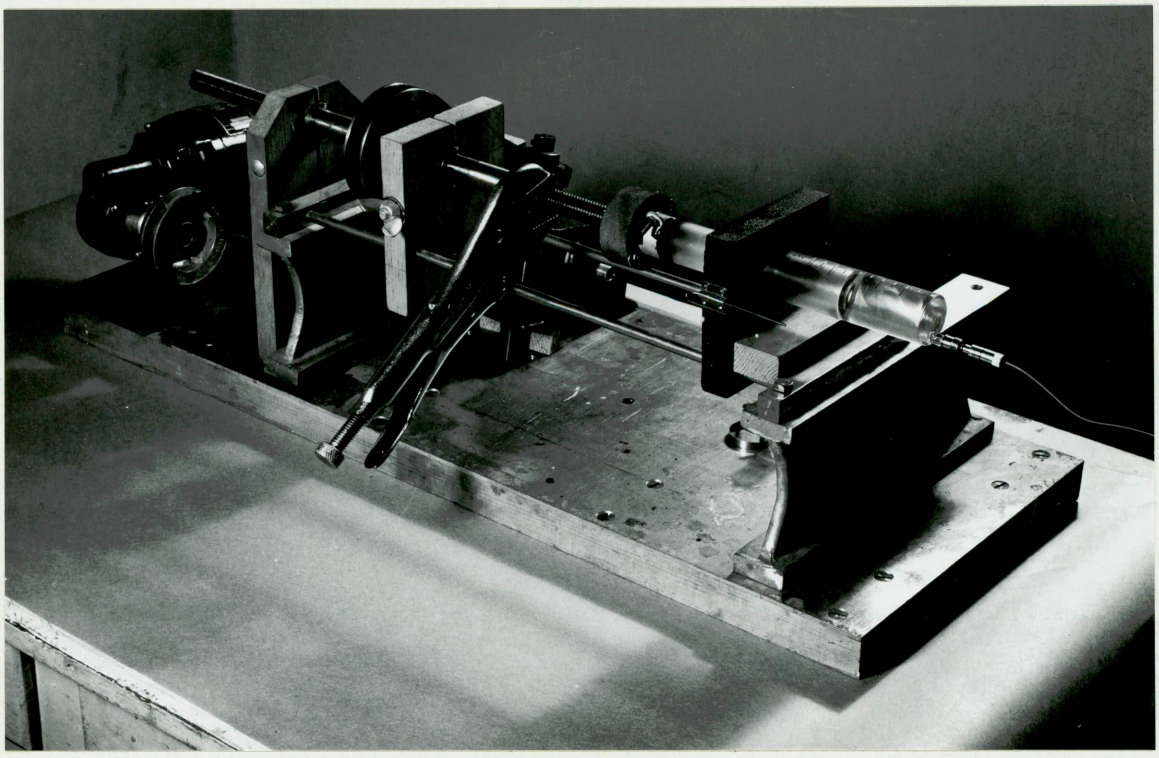

Figure I Oblique view of the arterial perfusion pump

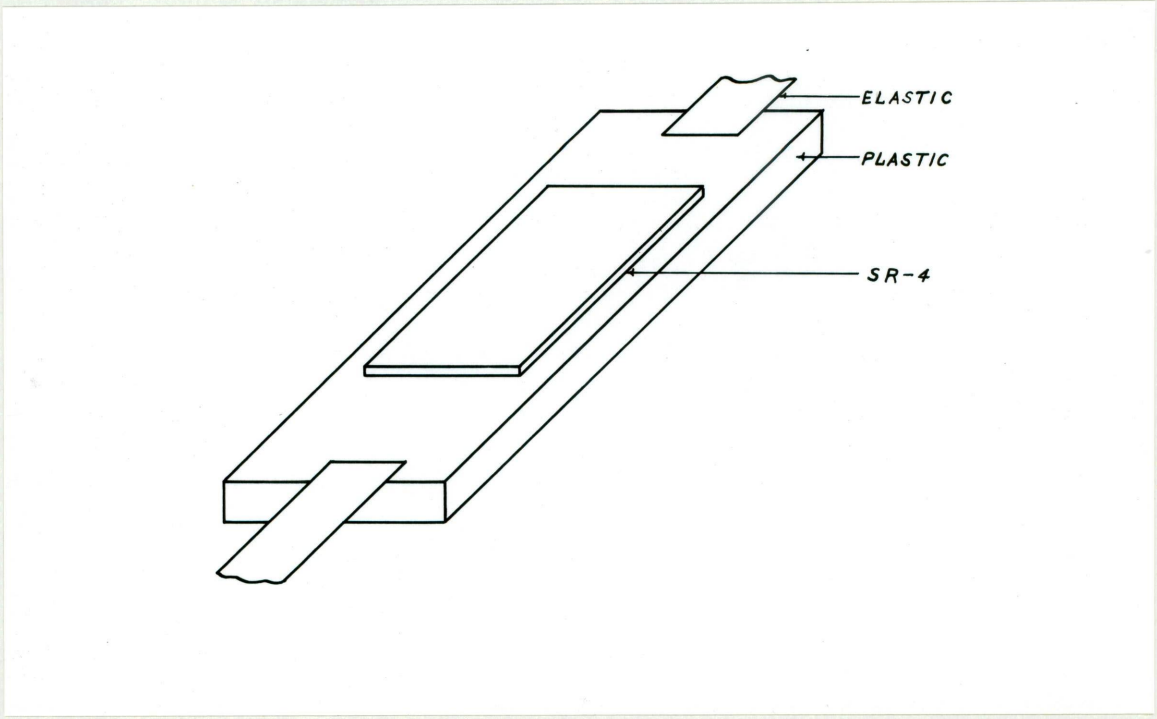

Figure II Semi-schematic drawing, strain gage pneumograph

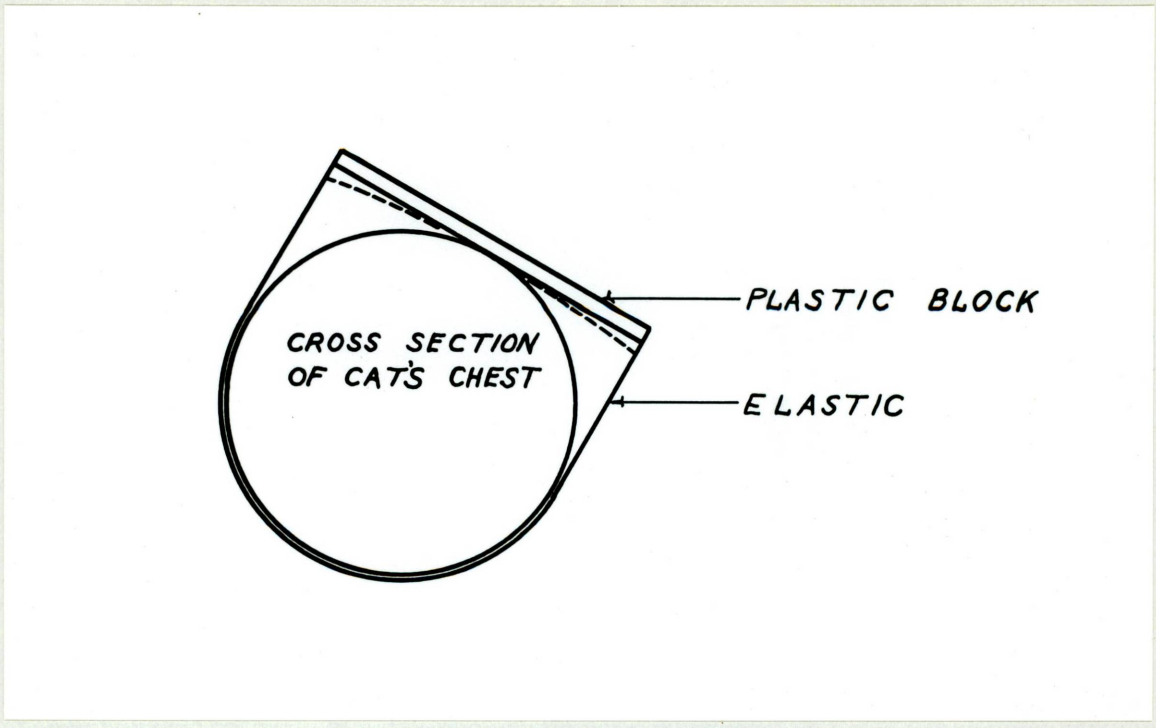

Figure IIa Semi-schematic drawing, strain gage pneumograph, dashed line is deflected position

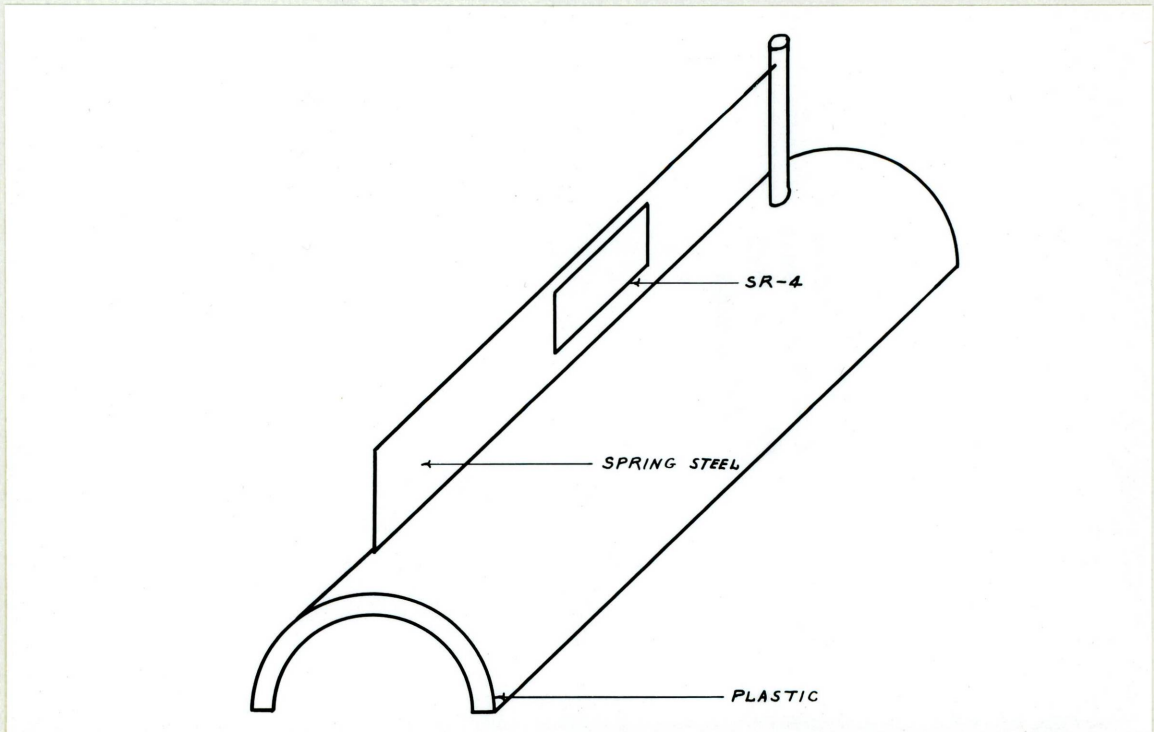

Figure III Semi-schematic drawing, modified strain gage pneumograph

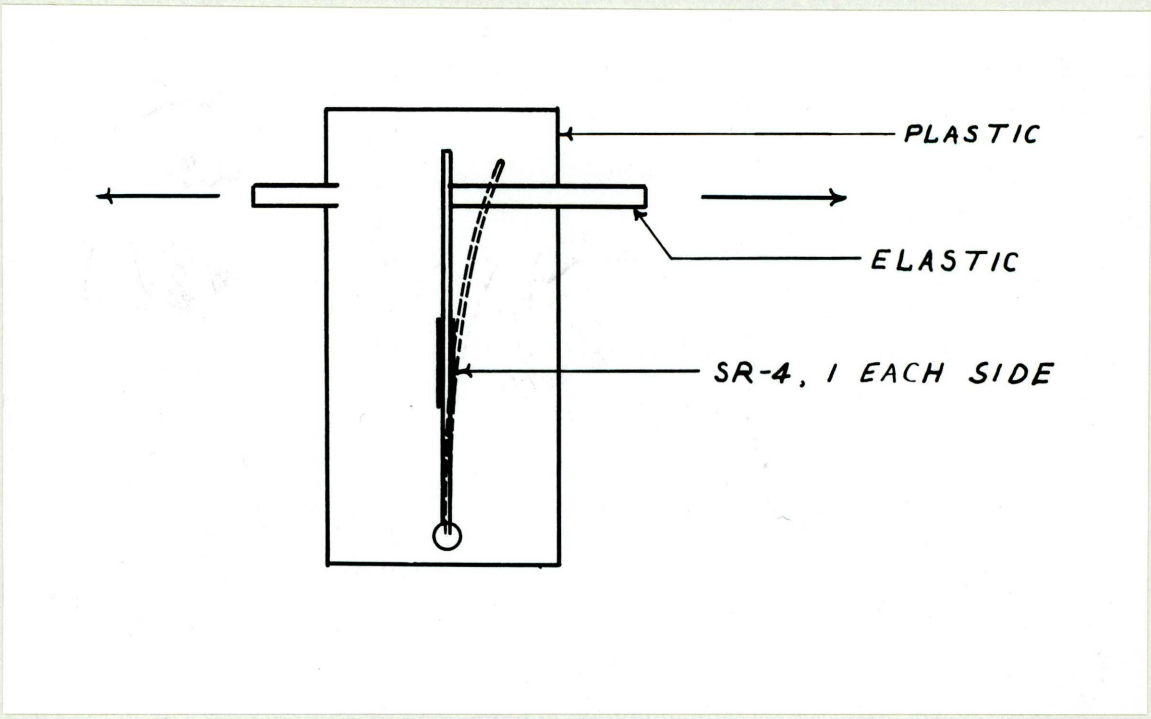

Figure IIIa Vertical view, dashed line shows deflection

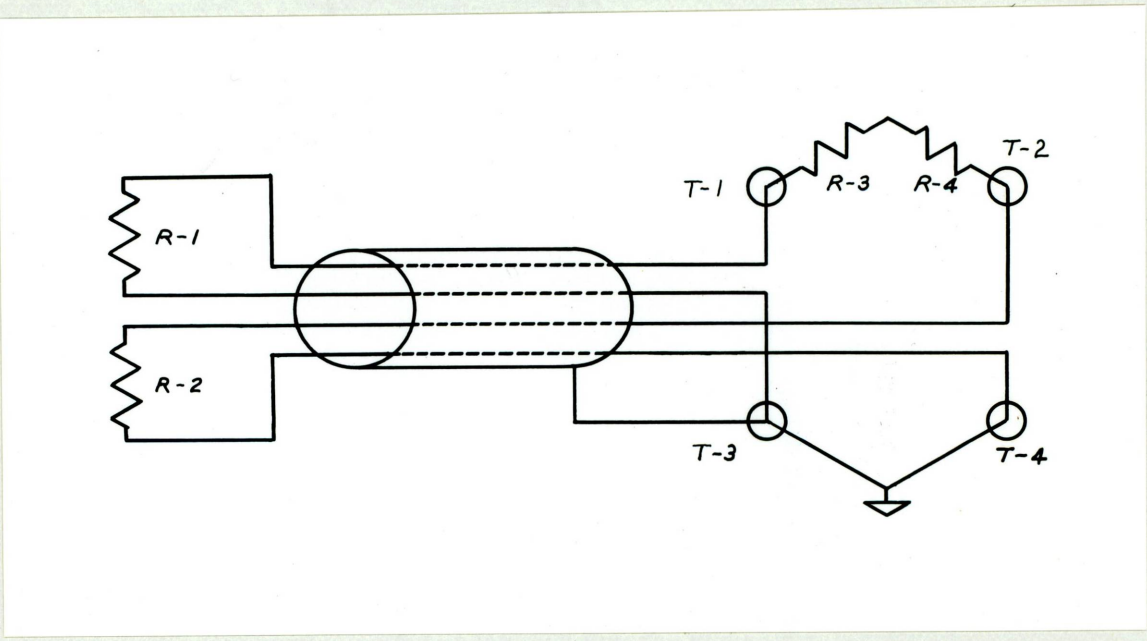

Figure IV Schematic wiring diagram, strain gage pickups. R-1 and R-2 are SR-4 strain gages (see text) R-3 and R-4 are part of preamplifier. T-1 through T-4 are terminals of the half bridge input.

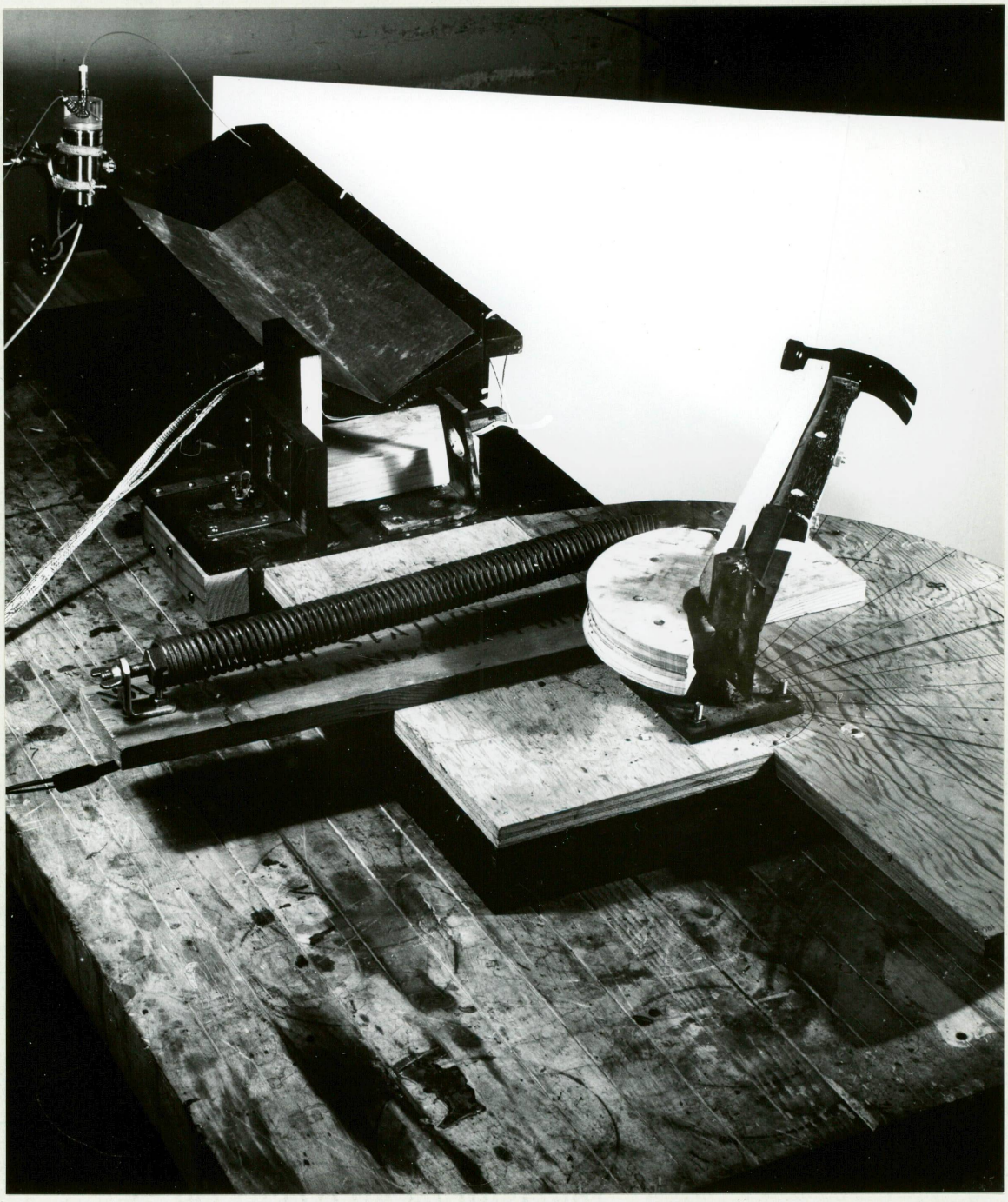

Figure V View of a spring loaded hammer

approximately two centimeters travel after contacting the cat's head, which was brought to rest after the blow in a cushion of folded paper towels.

The oscillograph has a built-in timer which inscribes a signal on the right-hand margin of the paper every second. In addition, a stopwatch was used in some experiments to measure time intervals in order to facilitate data reduction. The state of the reflexes and other pertinent data were written on the recording paper at the time of observation. Necropsies were not done because survival of the experimental animal or its lack was the principal goal in this work.

Because prolonged survival studies were not feasible, the ability of the respiratory mechanism to function upon the challenge resulting from the cessation of artificial respiration together with the state of the terminal arterial pressures were chosen as indicators of viability of the animal after severe head injury.

RESULTS

Some difficulty was experienced in obtaining prolonged respiratory depression with one blow. In about one-third of the experimental preparations it was necessary to employ more than one blow in order to achieve the desired duration of apnea. As was anticipated, duration of apnea was roughly proportional to the magnitude of the cerebral insult, which implies that this was apparently concussion as opposed to contusion and/or laceration which have somewhat different physiologic effects. Depending upon the response of the animal to artificial respiration the results can be grouped generally into four categories. Where more than one blow was necessary, only the last blow and the events subsequent to it were considered in the classification.

Group A consisted of six preparations in which spontaneous sustained respiration appeared after concussion induced respiratory arrest had been replaced by artificial respiration. The interval of time between the blow and the beginning of spontaneous respiratory movements range from 22 minutes to one hour and fifteen minutes with an average of about 50 minutes. Artificial respiration was employed only after the arterial blood pressure had dropped to just above one half its average resting value of 120 mm Hg. The elapsed time between the blow and the beginning of artificial ventilation ranged from about 2-6 minutes the average being approximately 3 minutes. Because of the hyperventilation produced by the respirator , return of function of the respiratory centers could be ascertained only after the challenge of disconnecting the pump at certain intervals. In no instance did spontaneous respiration result from the first such challenge which averaged 12 minutes after the blow. Because this time interval was never less than 8 minutes it appears that if artificial respiration had not been employed, death would have ensued. All these preparations save one were electively destroyed at intervals ranging from 1 hour, 48 minutes to 12 hours after the concussive blow. One preparation showed profuse hemorrhage from the nose and ears. This group did show prolonged survival on spontaneous respiration and it is believed that the artificial respiration was the important factor in maintaining life until spontaneous respiration could resume. The blood pressures at the time of elective destruction averaged above 90 mm Hg. except for the possibility of one instance in which the recording catheter was removed during the 12 hour survival at which time the cat appeared to be doing well. In another such instance the record was unfortunately misplaced or lost. Two of this series were given

neosynephrine to support blood pressure. In one case this apparently was decisive in survival inasmuch as the blood pressure did not rise after the use of artificial respiration alone.

Group B consisted of 12 preparations which showed no return of spontaneous respiration after concussion induced respiratory arrest in spite of the fact they were supported by artificial respiration. In three instances more than one blow was necessary to provide the necessary respiratory depression. The pre-concussion arterial pressures averaged the same as in Group A. The elapsed time of apnea averaged practically the same. If anything the arterial pressure had not dropped quite so much on the average, before the start of artificial respiration. Four animals died in a state of hypotension while being ventilated. Two of these were given maximum blows by the hammer after which their blood pressure dropped to low levels in a short time. Artificial respiration was terminated in the remaining eight preparations after variable intervals up to 6 hours after the cerebral insult. The average blood pressure in these eight was about 80 mm Hg. at the time of termination of the experiments. The average blood pressure at the termination of the experiment for all 12 preparations was about 60 mm Hg.

Group C consisted of six animals which showed either depressed respiratory excursion after the blow, or exhibited apnea followed by feeble respiration which in either case was inadequate to support the animal and therefore all were given artificial respiration. In all but one instance the blood pressure fell to 65 mm Hg. or less. Each animal except one, died as a result of cessation of artificial respiration, at periods ranging from 34 minutes to 5½ hours. One animal died in a state of hypotension in 15 minutes in spite of adequate ventilation.

Group D consisted of three animals in which feeble spontaneous respiration returned after a period of apnea ranging from a little less than 2 minutes to almost 2½ minutes, similar to those in Group A. Respiration either stopped or was inadequate and these three were supported with pump ventilation for variable periods of time ranging from 27 to 100 minutes. Each animal succumbed when artificial respiration ceased.

Inspection of the results in table A shows that one animal regained spontaneous respiration after one hour and 40 minutes and that this preparation lived for nearly three hours after resumption of spontaneous respiration. Because of this it appears that respiration might have returned in some of those not classified in group A if they had been given respiratory support for a longer period.

TABLE A

RETURN OF SUSTAINED SPONTANEOUS RESPIRATION AFTER CONCUSSION

Cat No.	Normal Blood Pressure mm Hg.	Duration of Apnea	Blood Pressure At Start of A. R. mm Hg.	Time Interval to First Challenge	Duration of Artificial Respiration	Life After Blow	Blood Pressure At Termination	Mode of Death
9	115	2 min.	62	8 min.	75 min.	2 hours 22 min.	128	Applied Asphyxia
16	137	3 min.	87	14 min.	60 min.	6 hours 57 min.	92	Sodium Pentobarbital
17	115	2 min. 16 sec.	92	10 min.	23 min.	1 hour 48 min.	90	Applied Asphyxia
22	127	2 min. 37 sec.	35	10 min.	47 min.	5 hours +	95	Sodium Pentobarbital
23	107	5 min. 55 sec.	65	20 min.	1 hour 40 min.	4 hours 20 min.	90	Later Record Lost
30	127	3 min.	56	10 min.	21 min.	12 hours +	Recorder Unhooked	Sodium Pentobarbital

TABLE B

NO RETURN OF SPONTANEOUS RESPIRATION AFTER CONCUSSION

Cat No.	Normal Blood Pressure mm Hg.	Duration of Apnea	Blood Pressure At Start of Artificial Respiration	Time Interval to First Challenge	Duration of Artificial Respiration	Blood Pressure At Termination	Mode of Death
1	120	2 min. 50 sec.	88	10 min. 20 sec.	14 min.	118	Discontinued Artificial Respiration
14	138	3 min. 20 sec.	18	Not done because of hypotension	14 min.	0	Hemorrhage And Hypotension
18	150	2 min. 30 sec.	95	Not done because of hypotension	77 min.	20	Hemorrhage And Hypotension
20	95	3 min.	50	11 min. 20 sec.	8 min. 20 sec.	103	Discontinued Artificial Respiration
21	133	3 min.	58	10 min.	4 hours 13 min.	58	Discontinued Artificial Respiration
25	128	3 min.	88	10 min.	2 hours 17 min.	57	Discontinued Artificial Respiration
Max. blow 27	133	1 min. 30 sec.	45	Not done because of hypotension	17 min.	33	Hemorrhage And Hypotension
28	118	2 min. 35 sec.	88	10 min. 50 sec.	31 min.	35	Hypotension
Max. blow 29	150	3 min. 5 sec.	81	10 min.	6 hours 15 min.	98	Discontinued Artificial Respiration
31	103	3 min. 30 sec.	90	10 min.	5 hours 15 min.	68	Discontinued Artificial Respiration

TABLE B
(Continued)
NO RETURN OF SPONTANEOUS RESPIRATION AFTER CONCUSSION

Cat No.	Normal Blood Pressure mm Hg.	Duration of Apnea	Blood Pressure At Start of Artificial Respiration	Time Interval to First Challenge	Duration of Artificial Respiration	Blood Pressure At Termination	Mode of Death
33	102	3 min.	68	11 min. 30 sec.	4 hours 12 min.	95	Discontinued Artificial Respiration
34	130	3 min. 30 sec.	80	32 min.	4 hours 4 min.	60	Discontinued Artificial Respiration

TABLE C

DEPRESSED OR IRREGULAR RESPIRATION, SUPPORTED BY VENTILATION EVENTUATING IN FAILURE

Cat No.	Normal Blood Pressure	Post-Concussion Respiratory Pattern	Blood Pressure At Start of Artificial Respiration	Artificial Respiration Started After Concussion	Duration of Life After Blow	Terminal Blood Pressure	Mode of Death
10	128	Respiration Slow	68	1 min. 32 sec.	62 min.	63	Discontinued Artificial Respiration
11	130	Respiration Feeble	80	6 min. 6 sec.	34 min.	65	Discontinued Artificial Respiration
12	125	Spontaneous Respiration Gradually Diminished	130	7 min. 30 sec.	37 min.	110	Discontinued Artificial Respiration
15	135	14 sec., apnea Respiration 6 min., 30 sec. Apnea	70	6 min. 30 sec.	91 min.	43	Discontinued Artificial Respiration
24	135	1 min.,20 sec. Respiration 4 min., 20 sec. Apnea	68	4 min. 20 sec.	5 hour 30 min.	45	Discontinued Artificial Respiration
32	118	1 min., 20 sec. Respiration 7 min., 20 sec.	23	7 min. 20 sec.	15 min.		Hypotension

TABLE D

RETURN OF SPONTANEOUS RESPIRATION AFTER CONCUSSION WITH SUBSEQUENT RESPIRATORY FAILURE

Cat No.	Normal Blood Pressure mm Hg.	Duration of Apnea	Blood Pressure At Start of Artificial Respiration	Time Interval To First Challenge	Life After Blow	Terminal Blood Pressure	Mode of Death
14	145	2 min. 24 sec.	88	15 min.	27	152	Discontinued Artificial Respiration
19	158	1 min. 55 sec.	43	15 min.	100	65	Discontinued Artificial Respiration
26	115	2 min. 26 sec.	58	6 min. 30 sec. Inadequate Spontaneous Respiration	56	38	Discontinued Artificial Respiration

DISCUSSION I

Kramer and Horsley (14), reporting in 1894, stated that death from gunshot wounds to the cerebral hemispheres was due to respiratory arrest. They used dogs under ether anesthesia and found that artificial respiration would usually prevent death. The effect on the respiratory center was shown to be due to pressure on the respiratory center.

In 1896, Kramer (15) reports, with a preparation similar to that used together with Horsley two years before, found a similar result. In this case a .22 caliber bullet (450 feet/sec., weight 32 grains) fired into a brass plate attached to the left parietal bone, or a falling weight was used to produce concussion. He found that the thorax remained in expiration during the apnea. He cites a typical "maximum effect" experiment. In this instance, apnea lasted 45 seconds and then 15 artificial respiration were made, after which normal respiration resumed, shallow at first and later becoming of normal character. The challenge period of 45 seconds is not sufficient, in our experience, to rule out spontaneous return of respiration. It is thus felt that this report presents no convincing evidence as to the effectiveness of artificial respiration. The blood pressure at no time reached a level below the pre-concussion state. He believed that this was a maximum effect and the immediate physiological effect of a blow was paralyzing the medullary respiratory centers.

Using cats under ether anesthesia, Cannon (2) found essentially the same thing as Kramer (14, 15) and Polis (18), i.e., that artificial

respiration could preserve life. Miller (16) agrees with Cannon, Kramer and Polis. He used dogs and cats under ether anesthesia. Walker, et al (19), stated that a minimal artificial respiration was usually needed to revive the animal and that recovery was "usually assured" if more vigorous artificial respiration was used.

Artificial respiration was found to be ineffectual by Denny-Brown and Russell (13) if the blow was of sufficient magnitude. This was not changed by an earlier onset of artificial respiration, etc. as stated above in the introduction. A delayed failure (after two minutes) of respiration is also reported. Some respiratory motion was noted after two minutes of artificial respiration, but late renewal of ventilation was ineffective. They also report failure of respiration after six minutes similiar to the one above. In all these preparations, there was no central nervous system damage as seen grossly and on microscopic sections stained with hematoxylin and eosin. Therefore these authors conclude that death is due to a loss of blood pressure with a mechanism similiar to surgical shock.

Webster and Freeman (20), using dogs under ether anesthesia, found that it was often necessary to use artificial respiration after 4 to 6 blows to the skull with a steel hammer, in order to preserve life until natural breathing returned. They were studying cerebro-spinal fluid pressure in concussion and made no other reference to respiration.

Gurdjian and Webster (8) agree with Denny-Brown and Russell that the mechanism of death is failure of blood pressure. However, they state that it is failure of the vasomotor center. Denny-Brown and Russell find that there is intense vasoconstriction peripherally and paralysis of the venous bed as in surgical shock. Gurdjian and Webster

used dogs under morphine analgesia. Their moderately injured preparations would at times show return of respiration coincident with improvement of rapidly failing blood pressure, but in the severely damaged preparation, artificial respiration was ineffectual in preserving life. Gurdjian and Webster (9) reaffirmed this position later.

In 1944, Olsen (17) stated "In this emergency (cerebral concussion) artificial respiration may save life". However, he presents no experimental evidence.

Groat, et al (5) reported on cats under chloralosane anesthesia. Threshold excitability of certain nuclei was studied with intracranial electrodes following concussion. These were concussed by a pendulum blow or by a weight dropped on a fluid column which was placed against the dura. Respiration was stated to be arrested in mid-position between expiration and inspiration. Apnea was observed to last 10 to 20 seconds, but in 7 of 28 cats artificial respiration was attempted and in 4 was successful in restoring normal respiration.

Dogs under morphine analgesia were concussed by a hammer blow by Gurdjian et al (9). They report one dog kept alive "only by giving artificial respiration for 7 minutes after the blow". This dog was sacrificed at 24 hours. In one other case artificial respiration was used for 5 minutes to prolong the dog's life. This report concerned primarily biochemical changes.

Groat et al (6) used artificial respiration to maintain concussed monkeys. Details of their use were not published. The primary purpose of this study was as in (5) above.

Gurdjian and Webster (7), using dogs under morphine analgesia, and employing a spring driven hammer similar to the one used in this laboratory, a pendulum or a hand held hammer, found that the blood pressure could fail with respiration intact. In others, temporary activity of respiration was closely associated with increased blood pressure. The use of artificial respiration was not reported. 150 dogs were used in this work, but the number losing blood pressure with respiration intact is not stated. This series is much larger than that being reported in this thesis, so it is more likely that a rare event would occur in it.

Brown, et al (1) report apnea lasting up to 50 seconds in concussed dogs with a spontanesou return of respiration.

Gurdjian and Webster (10) reported respiratory paralysis without spontaneous return and also respiratory paralysis followed by a return of short duration, followed by death. The severely injured animals showed failing blood pressure even when artificial respiration was going on.

DISCUSSION II

In the introduction above it is noted that the literature is not clear on the effectiveness of artificial respiration in the sustaining life during the apnea following cerebral trauma. This poses the problem as to what degree the vasomotor center is still active while the respiratory center is not functioning. The experimental work reported here demonstrates that often artificial respiration will maintain the animal for prolonged periods, with an adequate blood pressure, and also, that in some instances, if the respiration is maintained by artificial

means and provided the blood pressure is buoyed up by vasoconstrictors such as neosynephrine, some spontaneous respiration will return. The vasopressors were used in several cases, but their value is very questionable. They may help raise blood pressure for a short time, but high blood pressure may be a hindrance to spontaneous respiration as was seen in one preparation where a low blood pressure was maintained with fairly good natural respirations, but when the blood pressure was raised with a vasopressor, respirations stopped.

Six preparations (Table A) showed return of respiration after prolonged absence of respiration and its replacement by artificial respiration. In citing these cases as being "saved" by pump ventilation, the following assumptions are made:

1. During the initial apnea, the artificial respiration was not started before natural respiration might occur, i.e., not too early.
2. During the first respiratory challenge, sufficient time was allowed for the apnea of hypocapnia to have passed and the respiratory stimulus of hypercapnia to develop.

Both of these assumptions are valid in these experiments only in so far as judgement as to when to start the artificial respiration is valid. This was based on the rate of decline of the blood pressure and experience with similar preparations during the preliminary experiments. It is important to not push them over the limit which would result in almost immediate death, by too long withholding ventilation.

The time of relief of the initial apnea by artificial respiration is not as important evidence as is the first challenge to the respiratory center. In the six cited preparations the first challenges were from

8 minutes to 20 minutes after the blow. If, at this late time, natural respirations were not possible and the respiratory center was not capable of any respiratory drive, then it may, with reasonable confidence, be stated that this preparation would have expired if it had not been supported with artificial respiration. However, to prove death would have occurred in certain borderline circumstances, without death occurring, is difficult.

There is a large group of preparations which maintained a variable blood pressure after the trauma, but which did not at any time show spontaneous respiration. Some of these would have irreversible central nervous system damage from insufficient nourishment due to the prolonged low blood pressure. Others showed fairly high pressure at the time they were sacrificed by stopping the artificial respiration. These, aside from their direct brain damage, would be candidates for prolonged survival with possible return of natural respiration at a later time. That is, these might have recovered spontaneous respiration later if circumstances had permitted.

Then, of course, there are those in between, which may or may not sustain severe anoxic brain damage due to low blood pressure when artificial respiration is used. No evidence is herein presented as to long term survival. Certainly some animals could not survive regardless of the support given in these experiments. It could reasonably be assumed that some others (group A) may have recovered completely or nearly so. A large number fell in the middle ground and only a larger term experiment could shed significant light on this subject.

Four cases, clearly demonstrating loss of blood pressure with good ventilation are shown (14, 18, 27 and 28). External hemorrhage

being present, may or may not have played an important part in each, but the amount of blood lost could not be accurately measured. In any case, no amount of artificial respiration would support these preparations. It is possible, however, that something such as mucus may have plugged the airway, but this is not likely due to the short time involved.

SUMMARY

Twenty-seven experiments are herein reported in which cats were subjected to cerebral trauma as outlined above.

Six preparations showed prolonged apnea immediately after the blow and were sustained by artificial respiration until recovery of spontaneous respiration was achieved and life continued under spontaneous respiration more than one hour, forty-eight minutes later.

Twelve preparations did not show spontaneous respiration after the onset of apnea and were maintained on artificial respiration for varying periods. Four of these expired in spite of artificial respiration: all others were electively terminated.

Six preparations showed a transitory return of spontaneous respiration and its subsequent loss with maintenance of life by artificial respiration until elective destruction of the preparation. In one instance hypotension was marked.

Three preparations showed prolonged but inadequate spontaneous ventilation. These succumbed when the respiratory pump was disconnected at 27 to 100 minutes after the blow.

CONCLUSIONS

1. Evidence has been presented that artificial respiration may, in some instances of concussion, prolong life until the respiratory center may recover eventuating in the resumption of natural respiration, with several hours survival.
2. Some preparations may show prolonged survival with lower than normal blood pressure, but which cannot survive when artificial respiration is withdrawn.
3. Some preparations will expire with a gradual but steady loss of blood pressure while being well ventilated.
4. The variations in the literature reports are explained on the basis of the variable degree of physiological damage sustained by the animal. The early reports show evidence of a lesser degree of physiological damage than some later ones, and that some of alleged beneficial effects of artificial respiration may be questioned because of the ~~early~~ use of this form of therapy^(early) in the period of apnea.

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COLLEGE OF MEDICAL EVANGELISTS

School of Graduate Studies

ARTIFICIAL RESPIRATION DURING THE APNEA OF CEREBRAL TRAUMA

by

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An Abstract of a Thesis

in Partial Fulfillment of the Requirements

for the Degree Master of Science

in the Field of Physiology

May, 1958

Cerebral concussion often results in profound respiratory depression. The earlier workers in the field of experimental concussion concluded that artificial respiration could be a life preserving procedure in many instances. More recently, however, it has been held that this form of resuscitation is of little or no value.

Because of this conflict in viewpoint and inasmuch as the latter interpretation was made as a result of studies that were not primarily directed towards assessing the worth of artificial respiration during the apnea of experimentally induced concussion, this study was planned with the hope of resolving the differences of opinion.

A series of experiments was performed in which concussion, together with prolonged apnea, was induced in anesthetized cats by a spring loaded hammer while recording arterial blood pressure and respiration. When it appeared that the demise of the animal was imminent because of a rapidly falling arterial pressure, artificial respiration was provided in order to study the beneficial effects, if any, on the blood pressure and respiration.

In 6 instances out of 27 preparations, adequate spontaneous respiration returned. The arterial blood pressure was maintained at levels of 90 mm Hg. or higher for variable periods of time up to 12 hours after which the animals were electively sacrificed. Four preparations expired while being ventilated; in the balance demise accompanied the discontinuance of artificial respiration.

It is concluded that artificial respiration may be of value in the apnea of concussion in certain instances. In a majority of cases, however, it appears that when the blow is strong enough to cause profound respiratory depression, survival of the animal may not be anticipated.